BEDSIDE MEDICINE FOR BEDSIDE DOCTORS

An Open Forum for brief discussions of the workaday problems of the bedside doctor. Suggestions of subjects for discussions invited.

HYPERTENSION

WHAT CONSTITUTES HYPERTENSION

J. MARION READ, M. D. (490 Post Street, San Francisco).—How high must the arterial tension be before it becomes hypertension—140, 160, or 200? Even though we are discussing a quantitative increase in blood pressure, we cannot define hypertension by saying that all pressures exceeding a certain arbitrary limit are true hypertension. The subject is not so simple as that. For example, there is diastolic as well as systolic pressure to be taken into account.

Another point to consider is that hypertension is not a disease entity. We cannot name it on a certificate as a cause of death. Like leukopenia, hyperglycemia, achlorhydria, etc., it was nonexistent as a clinical concept until the technical means of measuring the deviation from an accepted norm was developed. Is it, therefore, entitled to the prominence it occupies in the minds of medical men? Would it occupy so prominent a place if it were not for the witchery of figures—and the feeling of power imparted by ability to measure a physiologic function and express it in numbers; an ability which in this, and many other instances in medicine, far outstrips our ability to interpret correctly the meaning of the figures after obtaining them?

The light in which hypertension is viewed today is analogous to that in which fever was regarded a century ago. We speak of hypertension in much the same manner that our predecessors discussed "the fever." Introduction of the clinical thermometer, about 1850, enabled Wunderlich, after eighteen years, to distinguish between various kinds of fever in his book "Medical Thermometry." The subject of arterial tension is more complicated than that of fever, and it has required twenty-five years of clinical sphygmomanometry before necessary distinctions have come to be recognized, and can be discussed as "bedside medicine."

In making some of the necessary distinctions, it is convenient to recall that, functionally and anatomically, the circulatory system consists of three parts—the heart, or motive force; the aorta and great vessels, or reservoir; and the arterioles, capillaries and veins, or distributing system. In discussing hypertension, it is convenient to keep these three divisions always in mind. In performing its function of maintaining an adequate blood supply, the circulatory system must frequently overcome handicaps interposed by disease in one of its three parts, or in other organs of the body. To accomplish this, an elevation of pressure is often the only mechanism available. Just as one view of fever regards it as a normal, beneficial response to infection, so we may also regard increased arterial tension as a physiologic response to disease somewhere in the body.

Whatever theory one may hold regarding hypertension, recent studies upon blood flow and the mechanics of the circulation make it necessary to revise some popular notions upon the subject. The manner in which our technical ability to measure arterial tension has been developed seems partially responsible for some of the misconception which has frequently marked discussions of hypertension in the past. I refer specifically to the fact that during its first decade clinical sphygmomanometry concerned itself solely with the systolic pressure. This point was emphasized and discussed in detail in this column over five years ago.* It is only necessary now to recall briefly that the diastolic pressure measures peripheral resistance and determines the pulse pressure necessary to maintain an adequate supply of blood for tissue nourishment; the magnitude of the pulse pressure being usually about half that of the diastolic pressure. Systolic pressure has no physiologic significance, being merely the sum of diastolic and pulse pressure. Since only that portion of the heart's contractile force exerted after the aortic valve is opened (*i. e.*, pulse pressure) is effective in propelling the blood through the vascular system, the body seems to employ various available mechanisms to maintain an adequate pulse pressure.

In defining and classifying hypertension, it is necessary to consider whether both diastolic and systolic pressures are raised, or if the systolic alone is elevated. A high diastolic pressure without a proportionately high systolic pressure is rare except in myocardial failure, although a low or normal diastolic pressure with elevated systolic pressure is frequently encountered.

It would clarify our understanding if the term "hypertension" were used only in referring to high diastolic pressure, since elevated systolic pressure occurs in various physiologic states simply as a result of increased pulse pressure, and in pathologic states where wide pulse pressures are compensatory. A brief discussion of one physiologic state, and of the pathologic conditions in which elevation of pressure is a compensatory mechanism adopted to enable the circulatory system to maintain an adequate blood supply, should serve to illustrate and assist in arriving at what we mean when we speak of hypertension.

First Type.—There is a group of conditions, all marked by normal or low diastolic pressures with wide pulse pressures and consequent high systolic pressures.

The normal condition of exercise is one of them.

* Read, J. Marion: Adequate Arterial Pressure, "Bedside Medicine," Calif. and West. Med., 29:102, 1928.

Another is thyrotoxicosis, in which persistent circulatory adaptations resemble those which are temporary in exercise, namely, fast pulse and wide pulse pressure, both responses to demands for increased blood flow.

The circulatory adaptations in both exercise and thyrotoxicosis resemble the response to injections of epinephrin, the secretion of which within the body perhaps accounts for the elevated systolic pressure seen in excitement, as upon a nervous patient's first visit to the doctor, or when being examined for life insurance.

Once in a long time a physician sees one of those rare cases of compensated, complete heart-block with very slow pulse and very wide pulse pressure, indicating increased systolic output. Only by this compensatory arrangement can such an individual maintain an adequate circulation.

Aortic insufficiency might be considered here, although the wide pulse pressure usually results from lowering the diastolic rather than elevating the systolic pressure.

There is another condition in this class which might be regarded as a special form of hypertension, since the lesion which causes it is in the arteries. I refer to the normal diastolic and high systolic pressures one frequently observes in individuals past middle life, often without symptoms or other evidence of serious cardiovascular disease. The aortic second sound is sharp and ringing, the brachials are tortuous, and there is other evidence of large-vessel sclerosis; the roentgen-ray reveals an elongated aorta, occasionally even calcified plaques, and at autopsy the aorta and great vessels are found to be sclerotic and rigid. Replacement of elastic tissue in the aorta, whose recoil normally forces blood onward during diastole, has resulted in a compensatory mechanism resembling that in aortic insufficiency. The pathologic lesion in both aortic sclerosis and aortic insufficiency render it impossible for a portion of the heart's kinetic force exerted during systole to be stored in the distended aorta as potential energy to be released by elastic recoil during diastole. Loss of this function, which is important in the normal circulatory system, is met by widening the pulse pressure and accomplishing all of the onward propulsion of blood during systole.

These examples will serve to illustrate conditions which have been called hypertension because of their elevated systolic pressure, but which logically do not belong in this category, unless one wishes to regard aortic rigidity and its necessary accompaniment of elevated systolic pressure as one form of hypertension.

Second Type.—Use of the term "hypertension," it seems to me, should be reserved for those cases in which the diastolic pressure is persistently high. This condition results when the peripheral resistance is raised by narrowing of the lumen in the smaller arteries and particularly in the arterioles. Nor does it seem necessary for the arterioles of the whole body to become diseased; a fibrosis of the small vessels of one organ seems sufficient to produce an elevation of diastolic, with the necessary widening of pulse pressure, such as one encounters in glomerular nephritis. The same adaptations of blood pressure have been thought to occur in fibrosis of the basilar vessels supplying vital centers in the brain stem. In eclampsia, increased intracranial pressure and other disease states, the same blood pressure reactions occur, but it only clouds the issue to speak of them as hypertension.

This leaves only the condition known as essential hypertension—which term recalls "the idiopathic fevers" of a half-century ago. Here the lesion seems to be primary in the arterioles themselves, increasing peripheral resistance and inducing the usual vascular response—elevation of pressure to overcome the resistance. What causes the arteriolar disease and increased peripheral resistance? Is it prolonged vascular spasm which eventuates in permanent reduction of the lumen? Or is the vessel wall damaged by a toxin of infectious or metabolic origin? Whatever the etiology, this condition, in the present state of our knowledge, would come closest to being true hypertension.

Summary.—1. There are numerous conditions in which elevation of blood pressure, usually the systolic pressure, is a feature. This increase of pressure is usually compensatory, or purposeful and often only temporary, and should not be called hypertension.

2. There is a type of hypertension marked by normal or slightly elevated diastolic pressure, but wide pulse pressure with resultant high systolic pressure. This occurs in elderly persons when the normally elastic aorta and great vessels become sclerotic and rigid.

3. Another type of hypertension is marked by high diastolic and systolic pressures, indicating increased peripheral resistance. This occurs in younger persons in whom the arterioles are severely damaged in a disease process of unknown etiology.

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PROGNOSIS

D. SCHUYLER PULFORD, M. D. (California State Life Building, Sacramento).—Two fundamental principles underlying hypertension should be kept in mind in a discussion of prognosis—(1) heredity, (2) type of hypertension.

1. *Heredity.*—In cases of similar type, one need not consult a cardiorenal specialist to prognosticate the fate of a hypertensive, though they die cardiorenal-vascular deaths. With high blood pressure patients the following dictum holds: "I would like to know more and more about what kind of a patient has a disease than about what kind of a disease the patient has." So it is with hypertension. Other things being equal, each type of hypertensive disease kills its bearer more or less according to what was written in the heredity book by the gens of his or her parents, and grandparents. Whether this be a tendency to obesity, endocrine imbalance, subnormal arterial system or unstable vasomotor mechanism, or a deficiency disease or what not, nevertheless a study of one's forebears shows that mendelian unit characters play an important rôle in hypertension even as in cancer, color, and creed.

2. *Types of Hypertension.*—Hypertension, like epilepsy, is a symptom, not a disease, and is due to many conditions, some known and others unknown. When these conditions are unknown, it is called "primary" or "essential," and when known, "secondary hypertension."

Longevity of a hypertension patient depends a great deal upon its type. A convenient classification is as follows:

Type 1. Primary hypertension (essential; idiopathic).
  (a) Malignant (Fahr).
  (b) Benign.
Type 2. Secondary hypertension.
  (a) Fluctuating or intermittent: Seen in emotional states of fear, anxiety, worry, or in women at the menopause.
  (b) Chronic: Occurring in such diseases as glomerulonephritis, urinary obstruction, toxemia of pregnancy, hyperthyroidism, aortic insufficiency.

Type 1. *Primary Hypertension.* — (*a*) Malignant Primary or Essential Hypertension: This usually comes on in early life, but may occur in the years over fifty. It invariably leads to rapid death from cardiac or renal failure, or both. Less often are there vascular accidents, as the vascular tree is not primarily faulty. No treatment seems to stay the tide of onrushing dissolution. Cardiac hypertrophy is extreme in the milder types and uremia is rapidly fatal in the more fulminating types. In this group come the early deaths, say from thirty to forty years of age, with a duration of symptoms at times of only a few months. A study of the eyegrounds shows papillo-edema, a neuroretinitis with sclerosis of the small retinal arterioles. This eyeground finding is a bad prognostic sign in hypertension. This often antedates uremic symptoms with which the patients die who develop a nephrosclerosis of high degree. Hyperemia of the optic disks and sclerosis of the smaller retinal arteries are seldom seen, or are only slight, in primary arteriosclerotic Bright's disease or glomerulonephritis.

(*b*) Benign Essential or Idiopathic Hypertention: This is an entirely different affair. Rest, restricted activity of mind and body, sedatives, specific depressor medicines and diet control the blood pressure's level, and these patients may, and often do, live out their normal life span, especially if they have inherited a fairly good cardiovascular system.

Renal risks of death are less apt than cardiac or vascular. In this group occur continued very high blood pressures which persist up to the sixth and seventh decades, or later with enormous hypertrophy of heart and normally thickened blood vessels.

Asymptomatic optimum blood pressure levels as high as 230/130 are often encountered with a history of carrying this pressure for years. The disease may persist for years without any important symptom and only be discovered during a routine physical examination such as examination for insurance. It is in this group that coronary narrowing often occurs, and it should be emphasized that inexpedient radical reduction of blood pressure by drugs is like "paving hell with good intentions."

In the essential hypertension group we have a cardiovascular hypertrophic change, and not a senile degenerative change with its tendency to cerebral hemorrhage and coronary infarction. If a short stay in the house "Casa Mana" and vasodilitants proves the power of arterial relaxation is still present, the prognosis is good. Transient or permanent paralysis is due to cerebral arterial thrombosis and does not often kill, and cerebral hemorrhage seldom takes place with diastolic blood pressures under 115. Death is usually at a late age from cardiac decompensation or intercurrent infections. The necessary adequate intra-aortic pressure being reduced allows coronary thrombosis or an ischemic myocardium and the reduced intercerebral pressure, cerebral thrombosis or anemia, none of which is apt to occur if the pressure is left comfortably high.

Even when hypertension cases are typed according to the above rather arbitrary classification there is a wide variation in individual longevity, so that perhaps we have in primary hypertension only one fundamental entity differing only in its speed of anatomic change and in the organ most attacked. Calling the difference heredity is a convenient cloak to cover our ignorance of the several unknowns involved.

One's idea of prognosis in primary hypertension as a whole may be clarified by a study of Dr. E. T. Bell's report of his series of four hundred and twenty cases of essential hypertension subdivided into five groups on the basis of post-mortem findings and the major clinical symptoms. In that series, the causes of death were given as follows:

Myocardial insufficiency accounted for	187 or 44 %
Coronary disease accounted for	67 or 16.4%
Apoplexy accounted for	81 or 19 %
Renal deaths accounted for	36 or 8.6%
Miscellaneous (accident and intercurrent disease)	49 or 12%

The renal group die younger and faster, so may be called malignant. The heart, coronary and cerebral group are, as a whole, more benign. The majority die myocardial deaths.

Type 2. *Secondary Hypertension.*—(*a*) Fluctuating or Intermittent Type: It is controversial whether this type is an entity or a prodrome of chronic secondary hypertension. A diagnosis should only be made on repeated examinations with diastolic pressure as well as systolic pressure increased, for emotional states such as excitement, fear, or worry will often temporarily raise systolic pressures—not so definitely the diastolic. Women at the menopause certainly seem to have temporarily increased blood pressure, which later subsides to have no effect on longevity. Younger people in this group are said to have some "vasomotor instability" and not to belong to the hypertension class; certain ones of them, however, should be suspicioned of going on to a permanent hypertension. Hyperthyroidism in its early stage should be ruled out.

The group, however, as a whole has a good prognosis. Hypertension from urinary obstruction, anemia, and toxic nephrosis should be classed with the fluctuating types, as the hypertensions disappear when the condition improves.

(*b*) Chronic Type: Chronic glomerulonephritis constantly gives a hypertension and a cardiac hypertrophy. We consider this a primary kidney disease, with secondary hypertension. Arteriosclerotic Bright's disease, which gives the so-called "primary contracted kidney," is associated with high blood pressure. Prognosis depends upon the type and extent of the kidney lesion and not the hypertension. Hypertension of hyperthyroidism is most often found in the toxic nodular group of goiter. Prognosis, of course, depends on the early recognition and proper handling of the goiter syndrome. It should be mentioned that general arteriosclerosis of aorta and peripheral arteries is not, as a rule, associated with hypertension.

No study of prognosis in hypertension is complete without a review of the records of insurance companies. Mortality among persons insured, and not insured, is comparable, and insurance statistics should, therefore, be of interest to clinicians.

The Actuarial Society of America and the Association of Life Insurance Medical Directors have a joint committee on mortality which reports from time to time on the effects of various medical impairments on the length of life of their policyholders, many companies accepting substandard risks at an increased consideration.

Insurance companies have perhaps the best statistics on what constitutes a normal blood pressure for American men. Their records of the percentage of increased mortality according to departure from average is interesting and exact.

One series of 707,000 normal men reported from twenty-six large insurance companies, with blood pressure readings from their thoroughly reliable examiners only, gave the following average normal blood pressures:

Age	Systolic	Diastolic	Pulse Pressure
20	120	79	41
30	122	81	41
40	125	83	42
50	129	85	44
60	134	87	47

The interesting statistics on just what becomes to hypertensives, and at what year of their disease, is very well shown by insurance companies' records. They die either cardiac, renal, or vascular deaths in a pretty high percentage; but whatever the cause, they die earlier than normal in direct proportion to increased blood pressure when averaged on a large group, and not taken as individuals.

The United States Census Bureau and Death Registry data, collected for years, give us tables of normal expected years of life from any attained age. For example, a man, age forty, all things being equal, on the average lives twenty-six more years or to age sixty-six. Given the one abnormal factor of a blood pressure of 160 systolic at age forty instead of the normal average blood pressure of 125 systolic, and he lives only thirteen years, or just half the normal man's expected time. Although not necessarily true of all individuals, given large groups of such and it is the true average expected and predictable result of hypertension.

In a large group of hypertension cases, deaths occur earlier than expected normals in direct proportion to increase of both systolic and diastolic pressures. For instance, if the expected mortality for average normal blood pressure cases is 100 per cent in cases with 5 to 15 points increase in systolic pressure, we find an increased mortality to 113 per cent. With diastolic pressures 5 to 15 millimeters above normal, we find 115 per cent. Systolic and diastolic blood pressures 5 to 15 millimeters below normal give a reduced mortality to 94 and 93 per cent, respectively. Systolic and diastolic pressures beyond 15 millimeters above normal produce a very rapidly increasing death rate. We see that the mortality increases rapidly with the increase of blood pressure over average, and that the average blood pressure is not the point of lowest mortality, because a systolic or diastolic pressure slightly below normal brings a more favorable mortality. No evidence is available regarding very low pressures.

The good effect of a low blood pressure seems to be best at the older ages. The pulse pressure has little significance compared to the systolic and diastolic pressures. The most favorable group is that with both diastolic and pulse pressures slightly below normal. It is interesting to note that increase of blood pressure with an increase of weight was found to be uniform, but slightly more definite with the older ages.

Summary.—In conclusion, then, we may summarize as follows:

Given a knowledge of the heredity background of a hypertension person and the type of blood pressure increase, and we have sound basis on which to estimate prognosis. We see primary hypertension, either malignant and rapidly fatal, or benign and of long life. We see secondary hypertension—acute, fluctuating or intermittent—well when the nervous or organic disturbance causing it subsides, and a chronic secondary type having a prognosis dependent upon the disease underlying it.

Less important, yet nevertheless practical points to consider, are the patient's environment and occupation, social status, and intelligence. These factors may decide the amount of control one has over nervous strain or activity of mind and body, which are more important than diet and medicines in estimating longevity. Reduction in weight is important, however, as blood pressure seems often to reduce concomitantly.

It remains to be seen what permanent effects dinitrophenol may have upon the organism as a whole, after long-continued use or multiple courses, before deciding if its use may modify our ideas about the mortality rate among the hypertensives.

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TREATMENT

JOHN C. RUDDOCK, M. D. (1930 Wilshire Boulevard, Los Angeles).—To treat hypertension we must, then, admit or presume that there is such a disease. If we presume that hypertension is an entity in itself, then any treatment instigated toward hypertension is logical.

Pathologists have been unable to determine the blood pressure of an individual, although they do

find many secondary evidences that have resulted from an increased blood pressure. Pathologists have also been able to discuss primary factors which should result in the clinical finding of a hypertension.

To presume that hypertension is an entity, in itself a disease, is wrong, because we know that increased blood pressure is secondary to a great many conditions.

In order to treat what is known as hypertension, one must determine the etiology of that disease before any treatment of a specific nature toward the increased blood pressure is logical. When there is an increase in the blood pressure, over the average normals, which has continued for any appreciable length of time, many changes occur in the body as a compensation for this increased pressure. These changes are insidious and slow, and, when complete, that increased pressure then becomes normal for that individual, and does not demand treatment. If we lower this pressure by means of therapy of any kind, the body again must go through a compensatory period for the new lowered level.

In order to outline treatment for cases of hypertension, one can divide the treatment into two classifications: First, search out and treat the underlying factor that has caused the hypertension; second, accommodate the patient and his life to the increased blood pressure so that it may become normal for that individual. Do not attempt to accommodate the blood pressure to the patient's life.

In searching out underlying factors in a case of hypertension, there is demanded of the clinician a thorough history of the present illness, a thorough past history of previous diseases, a thorough physical examination, in a search of a possible etiologic factor. There may be an early arteriosclerosis. There may be kidney pathology. There may be an endocrine disturbance. There may be nothing except hypertension.

However, arteriosclerosis certainly demands different handling than hyperthyroidism; and nephritis would certainly be treated differently than that case in which nothing was found.

An increase in the average blood pressure causes the patient to complain of various symptoms which may be attributable to the increased pressure itself. There may be headaches, excessive nervousness, pain in and around the heart, fullness in the head, dizziness, and other vague symptoms, that may or may not be constant; and upon physical examination, we often find a heart that is enlarged and hypertrophied, with forceful beats. These symptoms are not constant and usually disappear if the patient is put at rest. They are merely phenomena that are manifest before the body has fully compensated for the increased pressure. Most of these symptoms are disagreeable, and it is the thing that brings the patient to the doctor.

These symptoms should be alleviated by the various means that the practitioner has at his disposal. Acetyl-salicylic acid, pyramidon, are excellent measures for headache. Nervousness can often be controlled by the simple bromid mixtures. All of them can be alleviated by a rest in bed.

The blood pressure, however, will remain constant. It should remain constant, because it is the result of a compensation in order to overcome some factor.

We should keep in mind, however, that when a hypertension is present the safety factor for that individual has been lowered. We know there are certain pressures beyond which arteries will break. We know that pressure increases with heavy meals, exercise, excitement, and since the safety factor has been decreased, then these other factors should be controlled. The patient should not be allowed to eat heavy meals. He should rest frequently often and sufficiently. He should not be allowed to have excessive emotional strains.

We know that in an individual with a hypertension and very marked cardiac hypertrophy, heart failure will eventually occur, and that individual must then be treated for a decompensated heart in the same manner as any other decompensated heart, irrespective of the tension. The treatment, however, of this type of heart failure is not satisfactory, because we are dealing with the end-result and the response to therapy is very poor.

We know that individuals with a hypertension are subject to various intracranial circulatory accidents, such as cerebral thrombi or hemorrhage. There are no preventive measures unless we say that it is less apt to happen in those persons whom we put at rest mentally and physically, on small meals, in whom there are no emotional storms.

However, we must admit that these complications occur in persons who are put in this status as well as those who are more active. Individuals with a long-standing hypertension are often seen with acute renal failure. It is questionable in these cases whether or not the kidney was the primary factor in the hypertension at the beginning, or whether we are dealing with changes in the arteries themselves. It is a question which has been much discussed in the medical literature.

In order to sum up in a few words concerning the treatment of hypertension, it is essential that the hypertension be classified, that an attempt be made to determine the underlying factor before logical treatment can be instituted. Hypertension is a bogie. It is something "that will get you if you don't watch out"! It is the cause of a great many neurasthenics among our patients in their attempt to reduce a blood pressure which has become normal to them, even though it is above the average normals. All drug therapy instituted toward the reduction of a hypertension is wrong. Drug therapy should be instituted entirely to alleviate the symptoms that are present in association with the increased pressure. Treatment of complications is merely preventive, and the regulation of the individual's life to the blood pressure is essential, not the attempt to regulate the blood pressure to the individual's life.

There are certain general measures of treatment that can be instituted in all cases: 1. Rest. 2. Diet. (Light meals, and not any specific type of diet.) 3. Regulating the social status and emotional life of the individual.